not well established, but once established, I have never had the experience of having the group change. I believe the explanation of such changes will be found in errors in the laboratory work from inactive sera or imperfect technique. I frequently give transfusions immediately following operations for patients who are still unconscious from the anesthetic and have never experienced untoward effects. As a precaution, however, before each transfusion, the grouping of both the patient and the donor, as well as the cross-agglutination tests, is carried out.

CORONARY ARTERY DISEASE—AN ELECTROCARDIOGRAPHIC STUDY*

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DISCUSSION *by James B. Herrick, M. D., Chicago; William J. Kerr, M. D., San Francisco.*

CORONARY artery disease, because of its historical interest and its relation to angina pectoris, has been the subject of numerous reports throughout a long period of years. The older textbooks of medicine, published since the time of Jenner, John Hunter, and Heberden, have all contained eloquent descriptions of the major symptoms of angina of the chest. The more recent articles of Herrick[1,2] and Benson,[3] and the monographs of Mackenzie[5] and Allbutt,[4] have presented in detail the historical, clinical and pathological aspects of coronary artery obstruction and occlusion. Largely dependent on the clinical observations of American physicians the symptomatology of coronary artery disease has been so clearly defined that the diagnosis is now made and confirmed with considerable frequency.

THE CARDIOGRAM IN DIAGNOSIS

The aid brought by the electrocardiogram in the diagnosis of coronary artery disease is of recent date. Eppinger and Rothberger[6] first published records made by the hearts of dogs in which the muscle at the base of the left ventricle had been injured by the injection of a solution of silver nitrate. Smith,[7] a short time later, produced remarkable electrocardiograms in dogs by ligation of branches of the left coronary artery. Subsequently Pardee[8,9] published electrocardiograms made during an attack of angina pectoris and assumed that in his patient injury of the wall of the left ventricle by occlusion of a coronary artery caused the peculiar form of the electrocardiogram. The tracings published by each of these observers have essential characteristics of striking resemblance. In a recent report Pardee[9] adequately correlated and described these abnormal electrocardiograms and gave the name "coronary T wave" to the peculiar alteration of the terminal ventricular complex often associated with the coronary artery disease.

Willius[10] some years ago, in a purely statistical study, pointed out the grave prognosis associated with significant inversion of the T wave. Although he did not differentiate between simple inversion of the T wave and the characteristic coronary wave described by Pardee, he did state his belief that significant inversion of the terminal ventricular complex was produced, in all probability, by vascular degeneration of the obliterative type with involvement of the heart muscle. We may assume, therefore, that both significant inversion of the T wave and the peculiar coronary T wave are, in all probability, produced by myocardial derangements or degenerations dependent on occlusion of large or small branches of the coronary arteries or on varying degrees of obstruction of these arteries. In a minimal number of instances localized myocardial damage might result from other causes than vascular lesions, but except when experimentally produced, few such cases have been reported.

We have been interested in the specificity of the coronary T wave as described by Pardee, and for this study we have chosen all electrocardiograms which have shown a downward, sharply peaked T wave with an upward convexity of the S-T or R-T interval in all or any leads except lead III alone. When characteristic and marked in lead III alone, we are convinced of its serious significance if associated with other evidence of myocardial damage, but our post-mortem material is as yet too scanty to be used as evidence,

* From the Medical Service of the Los Angeles General Hospital.

TABLE 1—*Eight Cases. Diagnosis of Occlusion of Coronary Artery made Before Death*

Name	Age	Sex	Blood Pressure	Heart Size	Symptoms	Electro Cardiogram	Wa R	Autopsy
L. D.	48	F		Normal	Vomiting, shock and pain for two days.	See Fig. 4	0	Occlusion of left coronary. Infarction of myocardium.
C. H.	69	M	150/70	Enlarged	Pain, vomiting, dyspnea, shock.	See Fig. 2	0	Occlusion of left coronary. Infarction of myocardium.
H. H.	82	M		480 Gms.	Pain, heart failure.	Typical	0	Mural thrombos of left ventricle with embolism of left coronary.
H. E.	59	M	110/76	3.9-10.6 Orthodiagram	Pain, dyspnea.	See Fig. 3	0	Thrombosis left coronary.
G. B.	49	M	252/144	Large	Pain, dyspnea, heart failure.	Typical	0	Thrombosis left coronary.
F. W.	40	F	190/90	8.6 10.8	No pain. Gradual heart failure. Aortic insufficiency.	Typical	0	Occlusion of left coronary.
W. P.	66	M		Enlarged	Two attacks of angina with death in last one.	See Fig. 1	0	Old and fresh myocardial infarction. Left coronary occlusion.
E. M.	72	F	158/90	Large	Frequent anginal pain.	See Fig. 6	0	Occlusion of left coronary artery.

Note—In all figures abscissael measure 10-4 volts and ordinates 0.04 seconds.

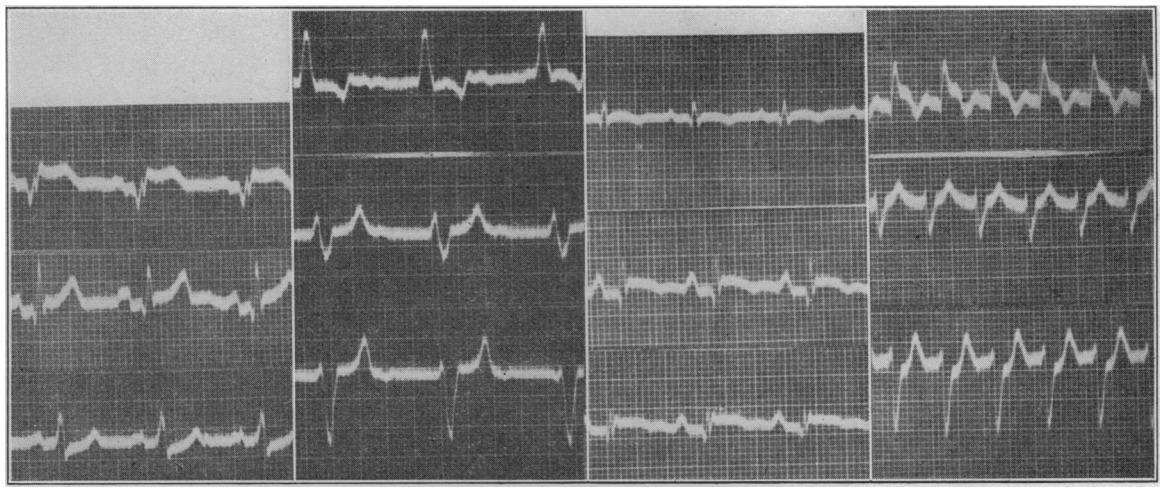

Fig. 1 Fig. 2 Fig. 3 Fig. 4

Figs. 1, 2, 3, and 4. Electrocardiograms taken during fatal attacks of angina pectoris. At autopsy an infarction of the myocardium from occlusion of a branch of the left coronary artery was found in each case. These figures show the characteristic changes of the T wave in recent infarctions.

and such instances were therefore not included in this report.

GROUPING OF THE ELECTROCARDIOGRAM STUDIES

The electrocardiograms were selected from approximately a thousand made at the Los Angeles General Hospital during the past year. They were then correlated with the clinical records in each instance and with the post-mortem studies in those cases which came to autopsy.

Statistics compiled in this way are of no value in demonstrating the interesting relationship between coronary artery disease and angina pectoris or heart failure. They are of value, we believe, in demonstrating the diagnostic value of the coronary T wave of the electrocardiogram.

The completed records fell readily into four groups, which will be discussed *seriatim*.

Group I: This group consists of eight patients in whom the diagnosis of occlusion of a coronary artery was made before death by clinical and electrocardiographic study and was confirmed by postmortem examination. In each instance the electrocardiogram (Figs. 1, 2, 3, 4), showed the

TABLE 2—*Eighteen Cases. Electrocardiograms, Histories, and Clinical Studies, All Compatible with Coronary Artery Disease Diagnosis*

Name	Age	Sex	Blood Pressure	Heart Size	Symptoms	Wassermann	Electrocardiogram
D. C.	65	F	212/110	Enlarged	Dyspnea. Edema; attacks of dizziness.	0	Fig. 5.
M. W.	59	M		Very large	Typical anginal attacks. Heart failure. Edema.	0	Broad Q. R. S. with slurring; coronary T leads I and II.
W. W.	58	M	110/84	8 - 13	Dyspnea; sudden attacks of weakness and tightness of chest.	0	Abnormal Q. R. S.; atrial fibrillation. Coronary T leads I, II, III.
F. P.	53	M	120/80	Normal	Attacks of angina for ten years.	0	Left ventricular preponderance abnormal Q. R. S. Coronary T leads III and T.
J. S.	50	F	155/100	Normal?	Frequent dizzy spells.	0	Left ventricular preponderance coronary T leads T, II.
T. B.	70	M	130/100	7 - 12	Marked cardiac asthma with anginal attacks.	0	Abnormal Q. R. S. Coronary T leads I and II.
L. L.	71	M	170/100	6.9 8.7	Anginal pain for many years.	0	Coronary T wave. Leads T. Very light upright T in lead III.
J. W.	41	F	110/78	5.4 7	Anginal pain.	0	See Fig. 7.
J. T.	64	M	136/80	5.3 13.8	Anginal pain.	0	Coronary T leads I and II.
A. S.	40	M	160/100	5.6 12.2	Anginal attack with pericardial friction. Death.	0	Coronary T leads I and II.
J. A.	54	F	250/130	Large	Attacks of weakness.	0	Coronary T leads I and II. Atrial fibrillation.
H. B.	52	M	212/150	Large	Anginal pain.	0	Coronary T leads I and II. Left ventricular preponderance.
M. H.	55	M	142/90	Large	Many anginal attacks.	0	Coronary T leads I and II; very high upright T in lead III.
M. S.	63	F	180/100	4.7 10.3	Many anginal attacks.	0	Coronary T leads I and II. Left ventricular preponderance.
N. G.	31	F	90/60	Normal	Attacks of breathlessness.	0	Coronary T leads II and III.
R. F.	54	F	200/100	Large	Anginal pain.	0	Coronary T leads I and II. Left ventricular preponderance.
G. S.	60	M	100/75	7.4 - 13.9	Many sudden attacks of dyspnea.	0	Coronary T leads II and I. [illegible] high upright T in lead III. Left ventricular preponderance.
C. K.	63	M		Large	Many anginal attacks.	0	Coronary T leads II and I. Very high upright T in lead III. Left ventricular preponderance.

characteristic abnormalities first described by Pardee in occlusion of a coronary artery in man. These patients all suffered from anginal pain (Table 1), and some had had numerous attacks of precordial pain. One patient was studied in two severe attacks of pain, and at autopsy a healed and a recent infarction of the left ventricle was found. Another patient with a history of a severe anginal attack previously was found to have an old mural thrombus in the left ventricle over an infarction and a recent embolism of the left coronary artery by a clot which probably arose from the old thrombus.

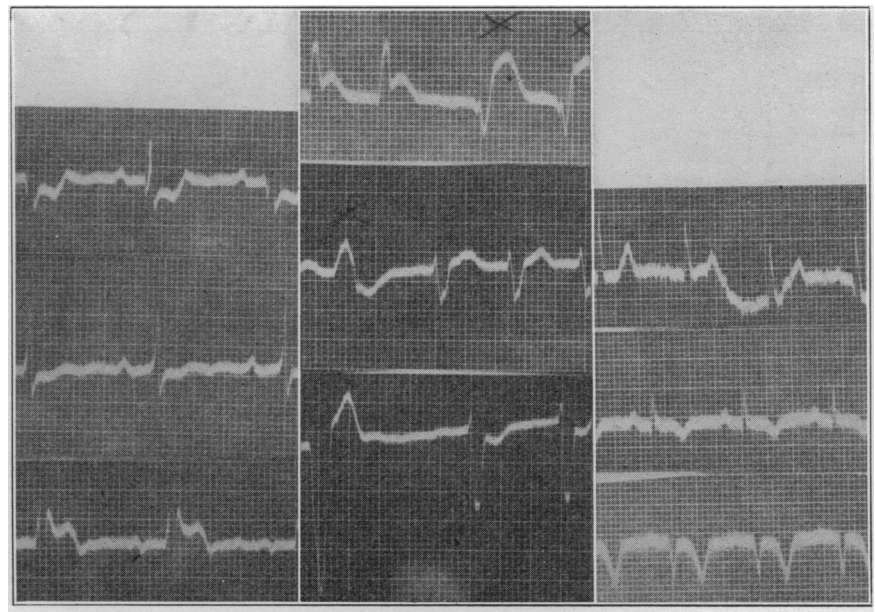

Fig. 5 Fig. 6 Fig. 7

Figs. 5, 6, and 7. Electrocardiograms taken during attacks of angina pectoris which were not fatal.

As is usual in each case the left coronary artery or one of its branches was occluded.

It is of interest that during an equal period of time thirteen patients, who had not had electrocardiograms made, died of occlusion of a coronary artery, unrecognized clinically, but demonstrated at postmortem examination. Among these was a second example of embolism of a coronary artery from a mural thrombus of the left ventricle.

Group II: We have placed in this group eighteen patients whose electrocardiograms were characteristic of coronary artery disease and whose histories and clinical studies were compatible with that diagnosis. Eight of these were females (Table 2). All of the members of this group presented evidence of grave heart disease. The majority complained of attacks of severe precordial distress usually associated with symptoms of heart failure. In the remainder, attacks of dizziness, weakness and breathlessness were considered to be the equivalent of actual pain. The electrocardiograms in this group (Figs. 5, 6, 7), in addition to coronary T waves, usually presented other evidences of grave myocardial damage, such as bending, broadening and slurring of the Q. R. S. complexes, left ventricular preponderance or bundle-branch block.

Group III: This group consists of nine patients with syphilis, syphilitic aortitis, and aortic insufficiency. All were males with positive Wassermann reactions except one whose blood Wassermann had been positive. The youngest was 38 and the oldest 68 years of age (Table 3). All but three had had attacks of anginal pain. All of these patients showed characteristic coronary T waves (Figs. 8, 9, 10), and all but one showed marked left ventricular preponderance by electrocardiogram. In a footnote to one of his articles Pardee mentions the occurrence of the coronary T wave in one instance of syphilitic aortic insufficiency,

TABLE 3—*Nine Cases. Syphilis, Syphilitic Aortitis, and Aortic Insufficiency Existing*

Name	Age	Sex	Blood Pressure	Heart Size		Symptoms	Wassermann	Electrocardiogram
S. C.	68	M	170/100	4.9	12.7	Aortic insufficiency. Anginal pain.	Positive	Coronary T leads I and II. Left ventricular preponderance.
W. S.	55	M	165/40	4.2	11.2	Aortic insufficiency. Anginal pain.	Positive	Coronary T leads I and II. Left ventricular preponderance.
A. V.	48	M	154/34	9	12	Aortic insufficiency. Aneurysm of arch. Sudden death.	Positive	Coronary T leads I and II. Left ventricular preponderance.
J. J.	43	M		4.2	11.3	Breathlessness in attacks. Aortic insufficiency.	Positive	Coronary T leads I and II. Left ventricular preponderance.
F. R.	47	M	140/60	8.9	11.8	Aortic insufficiency. No pain.	Positive	Fig. 8.
J. M.	39	M		Very large		Anginal attack. Aortic insufficiency.	Positive	Fig. 9.
J. E.	48	M	150/0	4.5	12.6	Anginal attack. Aortic insufficiency.	Positive	Fig. 10.
R. C.	48	M	130/50	7	12	Anginal attacks. Aortic insufficiency.	Positive	Coronary T leads I and II. Left ventricular preponderance.
R. G.	38	M	130/0	4.2	9.6	Severe anginal attacks. Aortic insufficiency. Has had positive Wassermann.	0	Coronary T leads II and III. Left ventricular preponderance.

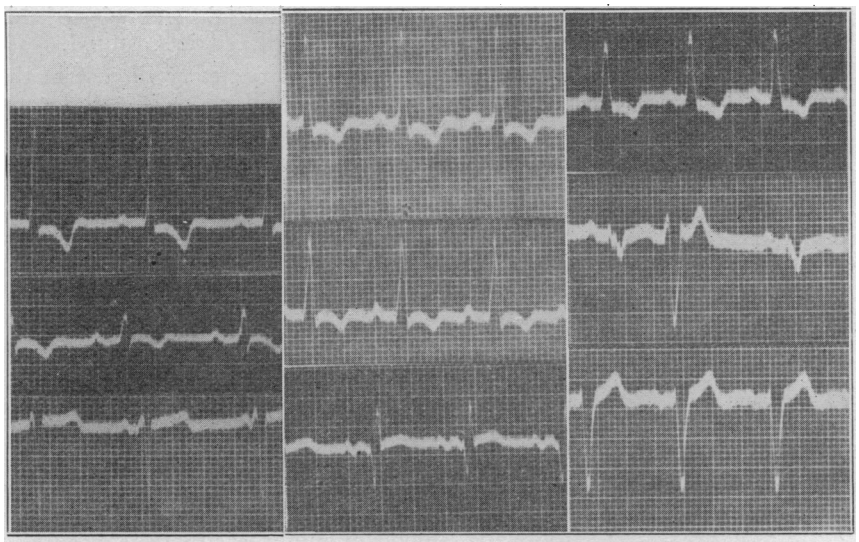

Fig. 8 Fig. 9 Fig. 10

Figs. 8, 9, and 10. Electrocardiograms from patients with syphilitic aortitis and aortic regurgitation showing coronary T waves and left ventricular preponderance.

but aside from that we have found no reference to it in the literature. The part played by coronary artery disease in the symptomatology of syphilitic aortitis is well known and the presence of the coronary T wave in a certain proportion of cases was to be expected.

Group IV: This group consists of nineteen patients of great importance in the study of the specificity of the coronary T wave. In each instance the electrocardiogram showed the characteristic coronary T wave, but clinical studies of the patient had not led us to suspect coronary artery disease (Table 4). We are aware that only postmortem examinations can prove, ultimately, the presence or absence of structural myocardial damage in the area irrigated by the left coronary artery in these patients.

CASE REPORTS

A summary of a few of the clinical records will be given:

CASE 1—J. H., 24-495. The patient, a male aged 60, was admitted to the hospital, comatose, with temperature of 104 degrees F. He had marked arteriosclerosis. The heart was said to be normal. The blood pressure was 185/120. Death occurred in a few hours. The electrocardiogram (Fig. 11) showed the abnormalities associated with coronary artery occlusion.

CASE 2—J. F., 262-338. A male, aged 52 years, was admitted for gangrene of the right toe, due to a circulatory disturbance thought to be thromboangitis obliterans. The heart was apparently normal. The electrocardiogram (Fig. 12) showed the changes associated with coronary artery obstruction.

CASE 3—C. G., 32-807. A boy, aged 19, was examined on account of asthmatic attacks occurring every three to four months. The heart seemed to be normal. The electrocardiogram is shown in Fig. 13.

CASE 4—A. T., 257-039. A male, aged 23 years, was

TABLE 4—*Nineteen Cases. Coronary Artery Disease in Doubt from Clinical Studies, with Electrocardiograms more Positive*

Name	Age	Sex	Blood Pressure	Heart Size	Symptoms	Wassermann	Electrocardiogram
H. B.	17	M	115/0	4.2 11.9	Severe anginal pain with breathlessness. Aortic and mitral endocarditis.	0	Coronary T in all leads.
L. K.	22	F	Normal	5.4 9.8	Severe anginal pain. Probably an embolus of coronary artery. Double mitral lesion. Death.	0	Figs. 17, 18, 19.
L. S.	18	F	Normal	Large	Acute febrile condition. White blood cells 16,000. Anginal pain. Probably a coronary embolism from double mitral lesion.	0	Fig. 16.
M. V.	24	F	Normal	5.1-8.6	Purpura fulminans. Death following hours of severe precordial pain.	0	Fig. 15.
L. S.	30	M	170/0	5.9-7.4	Acute endocarditis with aortic insufficiency. Severe precordial pain.	0	Coronary T leads II and III. Left ventricular preponderance.
N. M.	53	F	220/120	Large	Marked arteriosclerosis with dementia.	0	Coronary T leads I. Atrial fibrillation. Left ventricular preponderance.
L. M.	52	F	154/100	Normal	Diabetes mellitus. Diabetic gangrene.	0	Coronary T leads I. Intraventricular block.
R. K.	47	F	142/90	Normal	Diabetes Mellitus.	0	Coronary T in all leads.
H. B.	60	F		Normal	Myoma uteri.	0	Coronary T leads I and II.
A. T.	23	M	140/40	5-11	Severe anginal attacks. Aortic insufficiency. Double mitral lesion. Fever of short duration.	0	Fig. 14.
E. B.	30	F	120/60	Normal	Pernicious anemia.	0	Coronary T leads II and III.
C. G.	19	M	110/60	Normal	Asthmatic attacks every two or three months.	0	Fig. 13.
N. T.	60	F	160/110	Large	Diabetes mellitus.	0	Coronary T leads I and II. Very high upright T leads III.
J. W.	41	M	230/100	6.5 10.5	Heart failure.	0	Coronary T leads I and II.
D. H.	38	M	180/110	Normal	Chronic Brights disease.	0	Coronary T leads I and II.
J. O.	50	F	240/140	4.1 10.3	Arteriosclerosis.	0	Coronary T all leads.
J. F.	52	M		Normal	Gangrene of foot.	0	Fig. 12.
J. P.	63	M	150/110	6.5 11.3	Attacks of dyspnea.	0	Coronary T leads I, II, III.
J. H.	60	M	185/120	Large	Entered hospital in coma with fever; death.	x	Fig. 11.

admitted. Had fever and pain under the sternum and radiating into the left shoulder. He had a double mitral lesion, an aortic insufficiency and a pericardial friction rub was heard near the apex of the heart. All symptoms disappeared in a few days. The electrocardiogram (Fig. 14) showed a characteristic coronary T wave in each lead.

CASE 5—M. V., 259-007. A young woman, aged 24 years, entered the hospital with symptoms and signs of fulminating purpura hemorrhagica of two weeks' duration. She developed severe precordial pain, breathlessness and fever, and died in coma a few hours later. The electrocardiogram (Fig. 15) was taken shortly after precordial symptoms developed and showed a coronary T wave in all leads.

CASE 6—L. S., 253-627. A girl, 18 years old, was admitted on account of breathlessness and severe pain over the front of the left side of the chest. She had fever and a leukocytosis of 16,000. The heart was enlarged. There was a double mitral lesion and râles at each base. Her symptoms disappeared rapidly. Two months later, however, she complained that any unusual exertion was followed by severe precordial pain. Her electrocardiogram (Fig. 16) was characteristic of coronary artery disease.

CASE 7—L. K., 252-163. A woman, aged 22 years, was under observation for many weeks for grave heart failure. She had a double mitral valve lesion. She suddenly developed severe precordial pain and a pericardial friction rub at the apex of the heart. An electrocardiogram (Fig. 17) showed the curves of a coronary artery occlusion. Fig. 18 was made three days later and Fig. 19 six weeks later; shortly before death.

These abstracts are offered as evidence that in this group of patients coronary artery disease, or embolism of the coronary artery (Cases 4, 6), or hemorrhage into the myocardium or epicardium (Case 5) were probably causal factors in the abnormal electrical response of the left ventricle which produced the coronary T wave in the electrocardiogram.

COMMENT

The clinical and electrocardiographic records of these fifty-four patients are presented as additional evidence of the value of the coronary T wave in the diagnosis of coronary artery disease. In each instance in which post-mortem studies were made of a heart which had produced this wave, occlusion of the left coronary artery or one of its branches was found. Eight examples of this type were observed and placed in Group I.

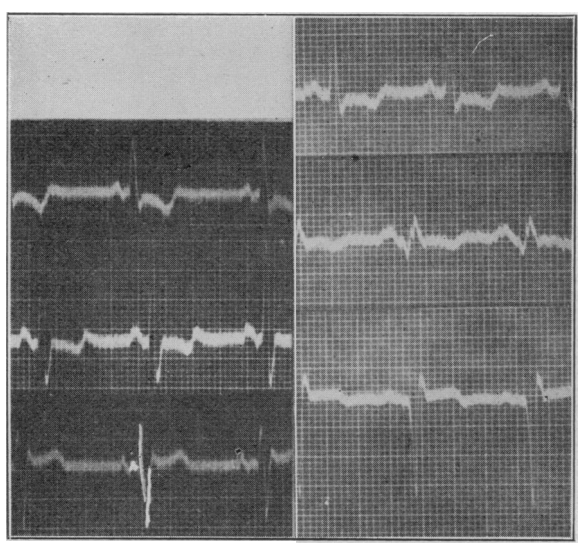

Fig. 11—Case Report 1 Fig. 12—Case Report 2

The presence of coronary artery disease in the twenty-seven members of Group II and Group III may be accepted with considerable certainty. These patients, as a rule, had anginal attacks and grave cardiovascular disease. Nine of the number had syphilitic aortitis and aortic insufficiency, and coronary artery disease under such circumstances is the rule.

Group IV consists of nineteen patients in whom coronary artery disease was not suspected until the presence of coronary T waves was observed in the electrocardiograms. Five of these patients were young individuals with valvular lesions of endocarditis and each complained of precordial pain. In these cases damage of the left ventricular muscle or of the left coronary artery by an inflammatory process or infarction of the muscle following embolism might easily have occurred. We have recently seen a fatal coronary artery thrombosis from a small inflammatory lesion in an otherwise healthy coronary artery. One patient in this group had a fulminating purpura hemorrhagica and developed severe precordial pain shortly before death. The remainder were past middle life. Several were diabetics with vascular degeneration and the others presented clinical evidence of cardiovascular disease. Anatomical proof of lesions of the left coronary artery or of degeneration from any cause of the area of the left ventricle supplied by this artery is absent in this group. Clinical and electrocardiographic evidence, however, practically prove the presence of a lesion of the left ventricular wall.

INTERPRETATION OF ILLUSTRATIONS

The illustrations in this article show the coronary T wave better perhaps than it can be described. In the examples of acute occlusion of a coronary artery the S-T or R-T interval, normally at the isoelectric line, may begin high up on the descending limb of the R wave. This feature is nearly constant in recent infarctions of the wall of the left ventricle. Later the commoner type of coronary T wave makes its appearance. With satisfactory clinical improvement even this abnormality may become less marked after weeks or months. Pardee believes that the type of T wave which appears at the time of infarction is probably produced by the reaction to the anemia of the heart muscle, while the coronary T wave may be dependent on the reaction of repair about the anemic area. We have seen a typical coronary T wave from a young person with pernicious anemia and also from a dying human heart just before the onset of ventricular fibrillation. It seems probable, therefore, that the abnormal electrocardiograms are the result of structural muscular damage from inadequate oxygen supply. That this condition is usually caused by obstruction or occlusion of an artery with subsequent temporary or permanent damage of ventricular muscle has been repeatedly demonstrated. The shape of the electrocardiogram is dependent, however, on the result of injury involving a certain area of the heart as demonstrated by Smith[7] and Eppinger and Roth-

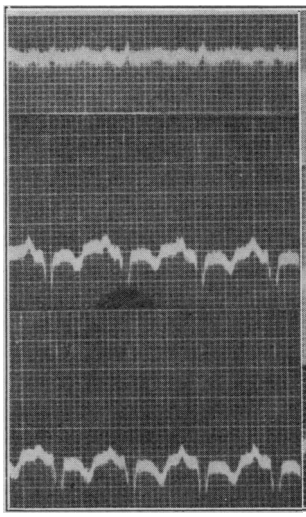

Fig. 13—Case Report 3

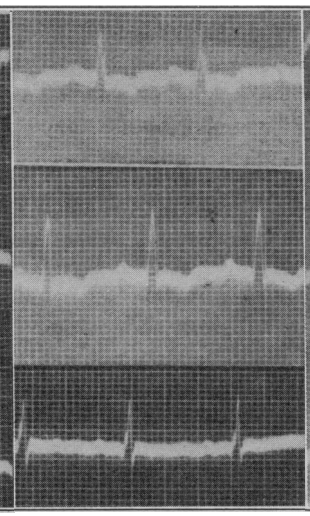

Fig. 14—Case Report 4

Fig. 15—Case Report 5

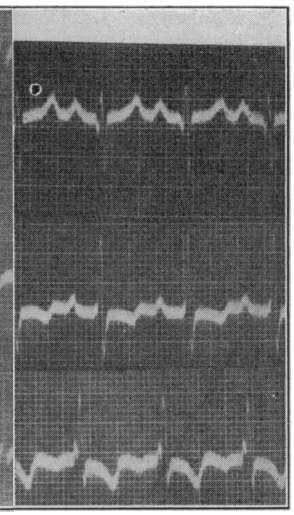

Fig. 16—Case Report 6

berger [6] on dogs, and Samajloff [11] on frogs. We have seen the same curves (Fig. 15) in the course of fatal purpura hemorrhagica and Cohn and Swift [12] have observed them in acute rheumatic fever.

COMMENT

Correlation of clinical electrocardiographic and autopsy studies of patients with coronary artery disease may amplify considerably the usual description of the symptomatology. In a number of instances the disease makes its first clinical appearance by sudden death. In the remainder, however, the symptomatology is extremely varied. This variability is an important diagnostic sign, and is produced by small areas of infarction due to obstruction or occlusion of smaller branches of the coronary arteries. These patients complain of attacks of dizziness after effort or sudden spells of breathlessness or palpitation of short duration often associated with substernal pressure or slight epigastric distress. Between attacks the patient may feel well, but minor symptoms may be found by careful questioning. Physical signs may be absent or, if present, may be difficult to evaluate. Sooner or later grave symptoms make their appearance. These are usually distress on effort or even severe anginal pain associated with symptoms of myocardial insufficiency. In a large number severe attacks of angina pectoris occur. In these patients the pain is often in the epigastrium and associated with fever, leukocytosis, and vomiting, and unless an apical pericardial friction rub is found, errors in diagnosis may be made. In the remainder myocardial insufficiency ensues and pain is never a prominent feature. The disease is produced essentially by slow sclerosis of the left coronary artery. When the artery is narrow in its proximal portion death from angina pectoris is the rule. When the thick-walled artery is dilated in its proximal portion multiple areas of myocardial degeneration and fibrosis occur from occlusions of small terminal arteries and left ventricular failure, often without pain, ensues.

CONCLUSIONS

1. The high degree of specificity of the coronary T wave has been confirmed by this study.
2. The presence of a coronary T wave in nine cases of syphilitic aortitis has been demonstrated.
3. Two examples of proved and two of probable embolism of the coronary artery have been recorded.

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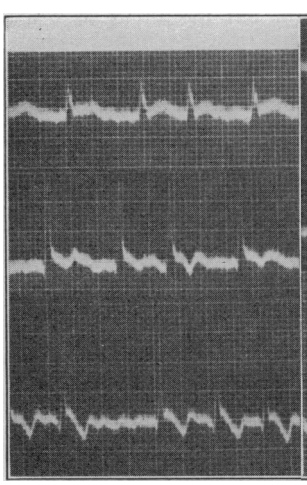

Fig. 17—Case Report 7

Fig. 18—Three days later

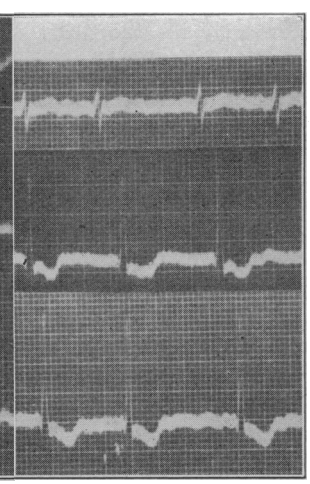

Fig. 19—Six weeks later

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DISCUSSION

JAMES B. HERRICK, M. D. (122 South Michigan Avenue, Chicago, Illinois)—Studies of this character are desirable, as they may throw new light on the physiology and pathology of the heart. They will also aid in diagnosis.

The clinical picture of acute obstruction of larger branches of the coronary arteries is fairly definite. Electrocardiographic findings are here confirmatory rather than decisive. They may, however, be helpful in telling the location of the lesion. The electrocardiograph may be of greater help in the group of cases in which there has been a sudden obstruction of smaller branches of the coronary artery, or in the other group in which the obstruction with resulting myocardial changes has been more gradual. It is in these two groups that the startling dramatic picture of sudden obstruction is lacking, and it is here that the tracing made by the instrument of precision may make clear symptoms that would otherwise be obscure. Such studies, therefore, as those of Doctors Berman and Mason are to be encouraged.

One must recognize, however, that an abnormal graph may be produced by lesions that are not in the strict sense coronary, at least not due to defective coronary circulation. Toxic conditions as met with in infections or in the free use of digitalis may markedly alter the electrocardiogram. Presumably here the effect is due to, primarily, alteration of the myocardium.

Perhaps the doctors' paper will convey the impression that it is by the changes in the T wave alone, or, chiefly, that coronary disease may be recognized, though I am sure they do not intend to convey such an impression. As their own figures show, other features also may point to disorder in the myocardium and the conducting system, *e. g.*, changes in the Q. R. S. portion of the graph.

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WILLIAM J. KERR, M. D. (University of California Hospital, San Francisco)—There is no doubt that the electrocardiogram may give us valuable additional information in regard to the condition of the myocardium. With such observations as we have made over many years, we feel that we can get a pretty good idea of what is going on in the heart muscle by electrographic study. The general picture of coronary occlusion with its persistent mediastinal pain, the appearance of vasomotor collapse, the slight fever, leukocytosis, and low blood pressure, is now becoming pretty well recognized by clinicians. This condition, I believe, should be sharply differentiated from the symptoms of angina pectoris with its transient attacks ushered in by effort and excitement and relieved by nitrites or rest. We have come to place a great deal of reliance on the general clinical picture plus the examination of the heart itself. On this examination of the heart we usually find a diminished first sound, which is often slurred or blurred at the apex, and a second sound which is greater than the first sound. Sometimes we find a pericardial rub in the acute cases. The x-ray film sometimes will show a bulging of the apex of the heart suggesting the possibility of an aneurism of the heart wall. This is more often seen in the old cases where there have been recurrent attacks of occlusion. The more one correlates the clinical findings with the electrographic findings the more one can predict what the electrocardiogram will show. The changes which we get in persons with a marked left preponderance, which is usually associated with hypertension, is of course well known to everyone. There is then a transitional stage which one can note bridging over to a rather typical bundle-branch block.

The usual criteria for an interpretation of a bundle-branch block should of course be present before we make such an interpretation, but we feel that we can anticipate the finding of a bundle-branch block. There are cases where defects in the conduction system produce bizarre electrical reactions. The higher we go in the Purkinje system the more sharply the lesions in the main branches of the bundle are differentiated, the more sharply the electrocardiogram will apparently demonstrate this. One, however, must be careful in his interpretation of the electrocardiogram, particularly in the direction and shape of the T waves in Lead 3. Sometimes also in Lead 1 the T wave may be inverted without other evidence of defect excepting for the presence of a left preponderance. Digitalis will of course at times confuse the picture, but in my experience with this drug, which agrees with the reports of others, inversion of the T waves in Lead 3 exceeds that of Lead 2 or Lead 1.

In referring to the electrocardiograms presented by the authors, I would place different and more complete interpretations on a great many of those shown. In Fig. 1, for example, there is no definite inversion of the T wave in any of the leads, although the T waves tend to be diphasic in Lead 1. There is definite slurring of the S wave which is also noted slightly in Leads 2 and 3. In this instance I would place the damage rather low in the heart and not involving one of the main branches. One certainly could not say here there was a typical T wave of coronary occlusion. In Fig. 2 there is a very marked left ventricular preponderance which is probably due to right bundle-branch block, showing all the necessary criteria for this interpretation. In such instances the T wave is opposite in direction to the main deflection. In this case there is also difficulty to make out the auricular waves and there probably is evidence of further damage in the auriculo-ventricular conduction system. In Fig. 3 the T waves are inverted in Leads 1 and 2 with the convexity upward, but the thing of great interest here is the small amplitude of the initial phase of the ventricular complex which, in our experience, indicates a grave myocardial damage. In Fig. 4 there is a definite tachycardia with a rate of about 150 with evidence of blocking in the right branch of the conduction system. This resembles very much some of the curves we have of ventricular tachycardia, although it may be due to a nodal rhythm. In these four examples, Figs. 1 to 4 inclusive, we have four entirely different types of electrocardiographic evidence of myocardial damage, and I do not believe that one could pick out any particular feature, such as the T wave, and say that that is the chief evidence of a certain kind of damage. In all instances there is evidence of grave myocardial damage, and in many of the examples shown, one might expect to find a patient with serious disease of the heart.

One might also take up some of the other figures shown, for example, Fig. 5, which shows a very marked left preponderance with what, I believe, is on the borderline between the ordinary left ventricular preponderance which we see and a right bundle-

branch block. There is also evidence of a delayed conduction time from auricle to ventricle in this case. Fig. 6 is one of the most interesting ones shown, and it seems to me there are so many variations in the complexes of the ventricle that we can believe that this indicates very grave myocardial damage. There are apparently extra systoles shown at "X" in the three leads with a very remarkable curve in Lead 1, suggesting ventricular tachycardia. There is probably a defect in the right bundle. No auricular waves are clearly shown suggesting that the patient has auricular fibrillation as well. I do not believe that the direction of the T waves in this case alone would give us any real clue of the cause of the trouble.

In Fig. 7 we have very marked inversion of the T waves in Lead 3 and less so in Lead 2. These we have seen in patients whom we have thought presented clinical evidence of a coronary occlusion, and I have not seen such marked inversion of the T wave in patients on digitalis therapy. In Fig. 8 we have another instance similar to Fig. 5 which is borderline between a left preponderance associated with hypertension or a high pulse pressure and that associated with a bundle-branch block. The T waves here are as characteristic as any shown by the authors for coronary occlusion. Figs. 9, 10, 11, and 12, show very much the same picture as Fig. 8. In Fig. 10, I believe, we can definitely say that there is a right bundle-branch block with one ventricular extra systole shown in Lead 2.

In Fig. 13 we see inverted T waves in Leads 2 and 3 suggestive of digitalis action. The rate, however, is more rapid than that usually observed when toxic digitalis effects have been produced. The same might be said for Fig. 16, which resembles Fig. 13 very closely. In Figs. 14 and 15 the T waves in Lead 1 are inverted and diphasic. In Figs. 17, 18, and 19, there is, of course, inversion of the T waves in Lead 3 and in Figs. 17 and 19 also of Lead 2. This could be due to digitalis therapy. In all instances the auricles apparently are fibrillating. I should not attach too much importance to the T waves in these cases in Leads 2 and 3 if they were not also inverted in Lead 1.

The authors' observations are very interesting, but I would call attention to the great variation shown in the electrocardiograms in these cases and wish to state that in my experience coronary occlusion may give a great number of manifestations all the way from slight evidence of myocardial damage to the more extensive type with complete heart block, or complete bundle-branch block, or ventricular tachycardia—even ventricular fibrillation. They might all occur one after the other in the same individual in different stages of the disease. The finding, however, of some of the bizarre forms which have been described, should help us to improve our knowledge of the underlying processes which produce the clinical picture in these cases. In many instances, I believe the evidence of grave myocardial damage can be found on the careful physical examination of the heart.

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Doctor Mason (closing)—We shall not attempt to discuss all the points brought up by Doctor Kerr in his comments. A few, however, need some clarification. None of the patients had received digitalis, so that factor may be dismissed. We were not interested primarily in "aborization" blocks, bundle-branch blocks, abnormal rhythms or evidences of myocardial damage in general, for such electrocardiographic alterations were to be expected and were found with great frequency in patients with coronary artery disease with or without angina pectoris. Nor were we interested primarily in inversion of the T wave. A study of the tracings published by Rothberger and Winterberger, Smith and Pardee, and of the tracings of proved myocardial infarctions in this paper, will show the changes in the S-T interval which are characteristic although possibly not pathognomonic of large areas of degeneration, from any cause, in the muscle irrigated by the left coronary artery. The S-T interval in such instances begins anywhere along the S wave, ending in a very acute T wave. This T wave is usually inverted in Lead 1, and high and upright in Lead 3. We feel sure that a study of these details will show the "family resemblance" of these curves. Doubtless many patients with myocardial claudication will not show such characteristic electrocardiographic curves, but that does not lessen the value of such tracings when present.

HALLUX VALGUS*

REPORT OF CASES

By Merrill C. Mensor, M. D.
San Francisco

Discussion *by Leonard W. Ely, M. D., San Francisco; Edward C. Bull, M. D., San Francisco; Maynard C. Harding, M. D., San Diego; John C. Wilson, M. D., Los Angeles.*

At the suggestion of Dr. L. W. Ely, who has for many years been interested in the possible congenital etiology of hallux valgus, I have attempted to collect and correlate some fifty-odd cases treated in the orthopedic clinic of Stanford University during the past three years.

The question of the cause of this deformity is still an open one and upon reviewing the literature we find that nearly every article presents a different theory. Perhaps this is one reason our treatment of this condition is not all that we desire.

REVIEW OF LITERATURE

In the early part of the twentieth century Dwight, and subsequently Young, of Philadelphia spoke of the congenital etiology of this deformity calling attention to the varus position of the first metatarsal and attributing it to an anatomical anomaly caused by an accessory bone between the first and second metatarsal which they designated as the "intermetatarseum." This bone mechanically forced the first metatarsal into varus, weakening the ligaments and causing the deformity. Subsequent workers have failed to identify this anomaly, and I have reviewed our x-rays very carefully as well as a series of some one hundred "normal" feet without finding this condition.

In 1912 Ewald called attention to the congenital etiology of this condition reporting a large series of cases. The facts which he brought out at that time are essentially as follows: there is present a definite varus of the first metatarsal with changes at either the metatarso-cuneiform joint or a twist in the shaft of the first metatarsal at its base which causes a varus deformity of this bone, with a secondary abduction of the phalanges. This picture reminds one of the structure of the foot in the ape and is probably evidence of an atavistic tendency. Over 50 per cent of these cases had a positive family history. Ewald very sagely questions that if this condition is due to ill-fitted shoes as advocated by a great number of physicians, why is it not more prevalent among the upper class of women who are constantly confining their feet in tight, narrow shoes? His conclusion tallies with my experience, that this deformity is far

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